



# Acute Compartment Syndrome

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## ABSTRACT

Acute compartment syndrome occurs most frequently in connection with injuries, terminal or chemical damage of tissues, ischemia, the activity of toxins or in patients with tissue ischemia or muscle necrosis. Clinical findings have found pronounced pain, followed by paresthesias, pallor, and paresis. Decreased pulsation of arteries has also been a frequent finding. In severe forms decompressive fasciotomy has been indicated within the first 12–24 hours after diagnosis. In the following paper, the authors present the case report of a 68-year woman who swallowed 1500 mg of trazodone as an attempt at suicide. After 12 hours her husband found her lying on the carpet with compression of the left arm under the trunk. The patient was treated conservatively and followed clinically, examined by ultrasonography, EMG and finally MRI.

## KEYWORDS

compartment syndrome; trazodone intoxication; ultrasonography; electromyography; magnetic resonance imaging

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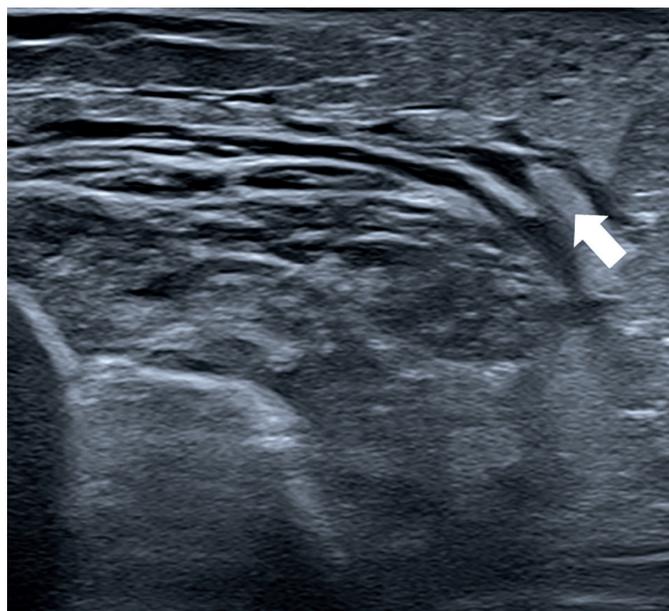
## INTRODUCTION

Acute compartment syndrome (ACS) is defined as an increase of intracompartmental pressure followed by perfusion pressure decrease with an ischemia of tissues in the compartment (1). Perfusion decrease develops into tissue necrosis with function impairment (muscle paresis, contractures of muscles), up to a loss in the extremities and, in exceptional cases, death. Most frequently ACS develops in connection with traumas (fractures, crush syndrome), combustion or chemical damage of tissues, infections (*Streptococcus*), compression from an overly tight fixation of extremity segments (plaster cast), local activity of toxins (snake bites), and after a longer compression such as from a disadvantageous position during surgery (lithotomic position) or in patients lying without moving for many hours due to a disturbance of consciousness or with intoxication (2, 3).

This paper details a woman with severe ACS after trazodone intoxication who was hospitalized by the authors.

## CASE REPORT

A 68-year woman was admitted to a neurological ICU with somnolence, oedema, pain, and plegia of the left upper extremity. The previous evening she had had a quarrel with her husband and in the morning her husband found her lying motionless on the carpet near the radiator with her left arm underneath her. Retrospectively she admitted, that she had taken 1500 mg trazodone as an attempt at suicide. She was first examined at the traumatologic emergency room where she received an x-ray of the spine and upper left extremity, CT brain, and CT angiography of cerebral vessels. The findings were normal. However, she had a very painful oedema of the left upper extremity, pain which increased upon touching the skin, severe paresis of all segments (no active movement of fingers and hand, very restricted flexion in elbow and abduction of arm) and a creatine kinase 71 ukat/l (0.35–3.58), ALT 3.06 ukat/l (0.10–0.78), AST 5.26 ukat/l (0.05–0.72) which is characteristic for ACS. The surgeon did not decide to perform a fasciotomy. The patient was admitted to the neurological department and treated conservatively including rehabilitation. An ultrasonography of the left arm and forearm showed oedema of muscles with an infiltration of tissues while arterial stream courses in the brachial and radial arteries were normal (Fig. 1). Over the course of 3 days the movements of the upper left extremity appeared and improved stepwise. An electromyography (EMG) on day 6 after admission bore evidence for ACS. The compound



**Fig. 1** Ultrasonography: Oedema and infiltration of left forearm.

muscle action potential of the median and ulnar nerves showed low amplitude and sensory nerve action potential for both nerves was only slightly lower (Tab. 1).

**Case history.** The patient has been treated for 15 years for asthma bronchiale, 10 years for hypothyreosis (Letrox 75 ug a day) and for depression (escitalopram 20 mg). Four years ago she cut the skin of her left wrist in a suicide attempt but without serious injury and without sequel.

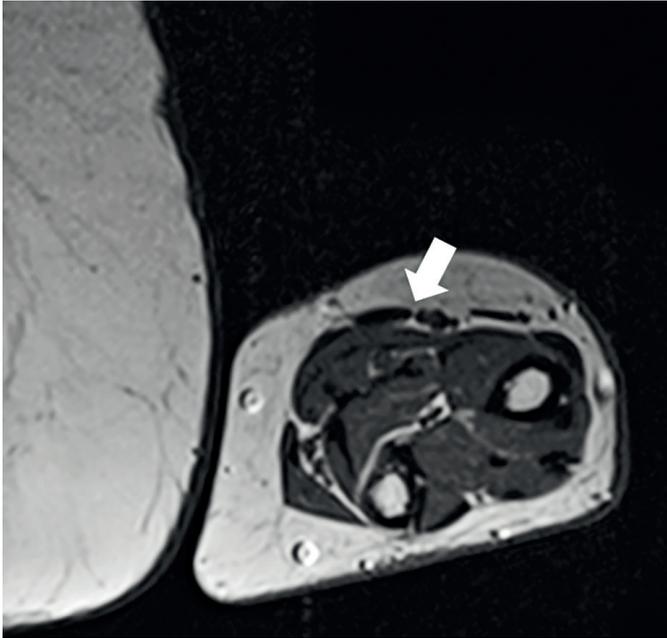
During the patient's stay in the department of neurology her CK level initially increased (3rd day 248 ukat/l) and after 10 days it dropped to normal values. The patient successfully trained with the help of a physiotherapist. A psychiatrist evaluated her problems as a depressive disturbance with a reactive use of trazodone. After an 18 day stay in the hospital she was dismissed with a prescription of trazodone 150 mg in the evening, vortioxetine 10 mg, hydroxyzin 15 mg in the evening and her regular daily medication. She stayed in the rehabilitation institute for 6 weeks.

After her final check 3 months after ACS she was substantially improved - able to button up and eat with a knife and fork. The handgrip of the right hand was found to be 46 and 10 kPa in the left (normal value > 40). However, she still showed weakness of arm abduction, external rotation, wrist, and finger extension but was able to pinch her fingers together. No muscle changes with shortenings or contractures were found. Hypesthesia was found only on the dorsal aspects of the wrist, thumb, and index finger. An EMG showed motor and sensory conduction studies of

**Tab. 1** EMG findings.

Muscle	Insertion activity	Fibrillation / Sharp waves	Frequency of MUP / amplitude	Tissue fluid
M. deltoideus sin.	Very low	0	0	Slowly leaks
M. biceps br. sin.	Slightly longer	0	3 / 0.6 mV	Leaks out
M. extensor digit. com. sin.	Prolonged	0	0	Leaks under pressure

MUP – motor unit potential



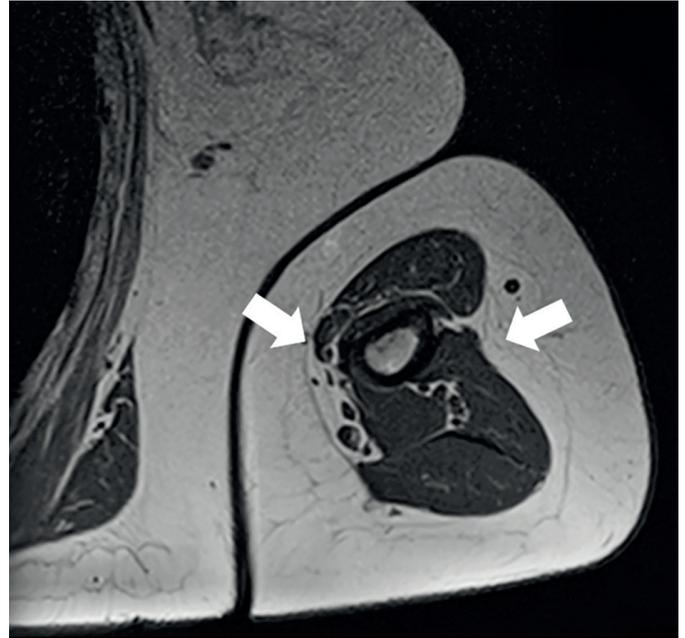
**Fig. 2** Axial MRI T1 weighted: Atrophy of forearm muscles.

nn. median, ulnar, radial to be normal. During a needle EMG investigation fibrillation, positive waves, small and short-duration MUP were found in all three muscles myotonic discharges. An MRI found atrophy of the muscles of the upper left extremity (Fig. 2, Fig. 3).

## DISCUSSION

ACS develops due to tissue pressure increase in a space tightly surrounded with fascias in the compartment. Because of the vasodilation of arterioles and an increased permeability of vessel walls an increased plasma filtration occurs. The increased pressure in the compartment and subsequent collapse of veins creates a decrease in tissue fluid resorption. In every segment of the extremities there is a direct number of compartments. In the arm there are 2 compartments, in the forearm 3 and in the hand 10. The most frequent ACS occurs in the forearm, and according to bone fracture in 35% of cases due to pharmacological/drug depression of consciousness with a compression of tissue in 10%, and in paravenous application of medicaments 8% (4-6). Here the patient had a pharmacological decrease of consciousness during the time she was lying on the carpet near a radiator with her left arm under her trunk. Our patient suffered of acute compartment syndromes of her forearm, arm and shoulder, but the posterior forearm and the anterior arm were the most affected compartments.

In English literature there is a "rule of 5P" for ACS. This rule includes pain (out of proportion), paresis, pallor, paresthesias and pulseless arteries (3). The next step for diagnosis is measuring the tissue pressure - if the pressure in a compartment exceeds 30 mm Hg then fasciotomy is indicated. This should be done during the first 6 hours following diagnosis. The next possibility of diagnostics are the neuroimaging methods. In our patient there were clinical findings typical for ACS. Severe left upper extremity paresis made it necessary to exclude stroke. A brain CT and CT



**Fig. 3** Axial MRI T1 weighted: Atrophy of arm muscles.

angiography did not find stroke or any other brain disorder. Tissue pressure was not measured. An ultrasonography of the arm and forearm disclosed tissue oedema but with a patency of arteries (5). Because of a long delay of admission (more than 12 hours since the beginning) and patency of arteries, the surgeon did not decide to perform a fasciotomy.

Electromyographical investigations on day 6 after admission showed a substantial decrease of compound muscle action potential for the median, ulnar, and radial nerves and slightly lower sensory action potential for all 3 nerves. In a needle EMG of 3 muscles pathological spontaneous activity was not found with an increase in insertion activity, no voluntary activity (MUP) in m. deltoideus and m. extensor digitorum communis and only 3 MUPS of normal parameters were found in m. biceps brachii. In a needle mark the tissue fluid leaked under pressure in m. extensor digitorum communis. The findings were characteristic for compartment syndrome with not severe muscle fibers damage.

At a check up after 3 months a significant increase of muscle strength was found. Muscle atrophy in arm and forearm was only moderate, hypesthesia persisted only for the radial nerve. In an EMG fibrillation and positive sharp waves, short and low-amplitude MUP were found, as well as myotonic discharges as an EMG parameter of direct muscle membrane damage. Muscle atrophy was found on the MRI. There were no muscle contractures and no deformities of the upper left extremity.

## CONCLUSION

Acute compartment syndrome develops on the basis of an increased pressure in the compartment followed by the breakdown of tissue perfusion leading to the development of necrosis. Diagnosis is determined based on clinical findings measuring tissue pressure in the compartment. A fasciotomy is recommended for ACS meeting these criteria during the first 6 hours (7). Ultrasonography and MRI are

important as complementary investigations. EMG contributes to the diagnosis with the evaluating grade of muscle fibre damage and the evidence of damage of nerves passing through the compartment.

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